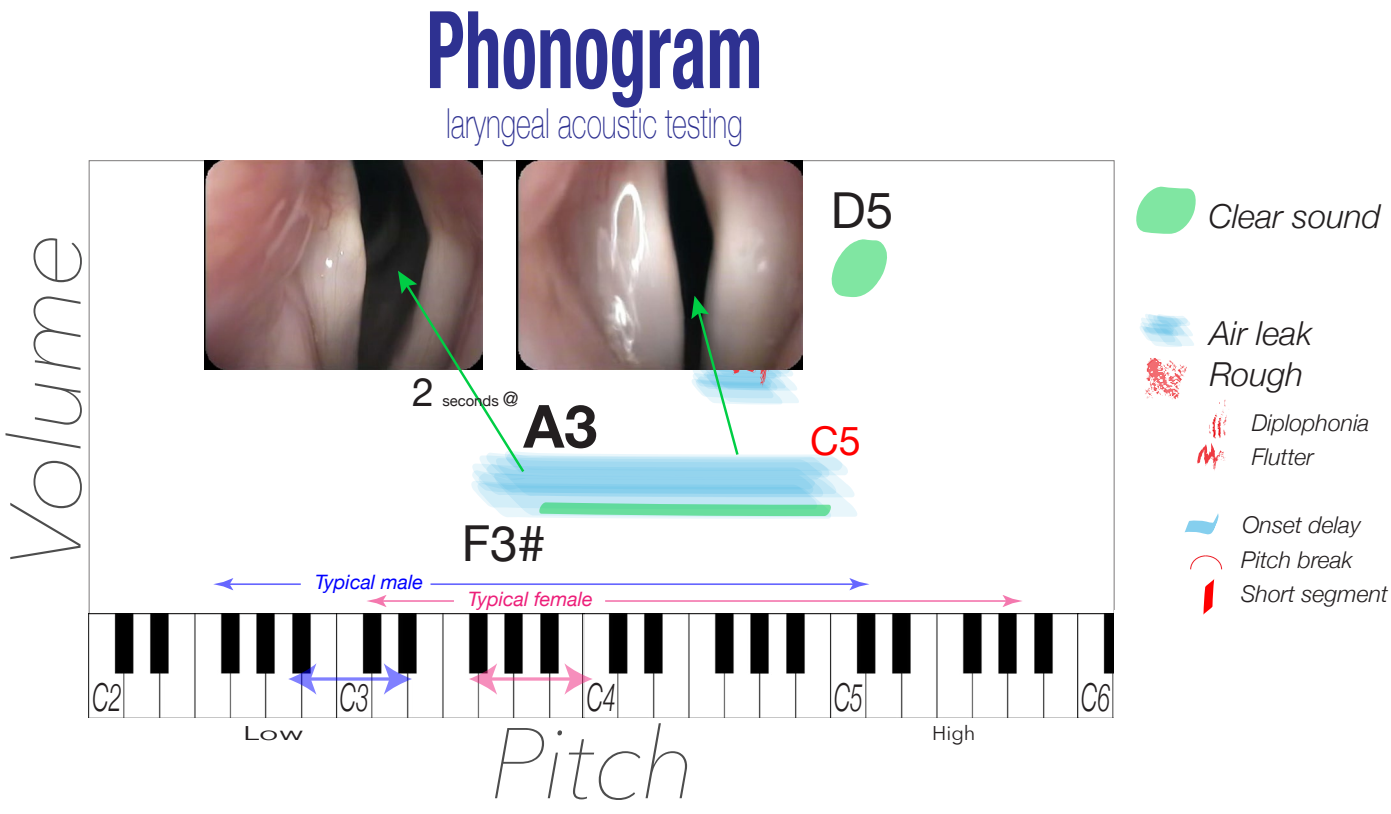
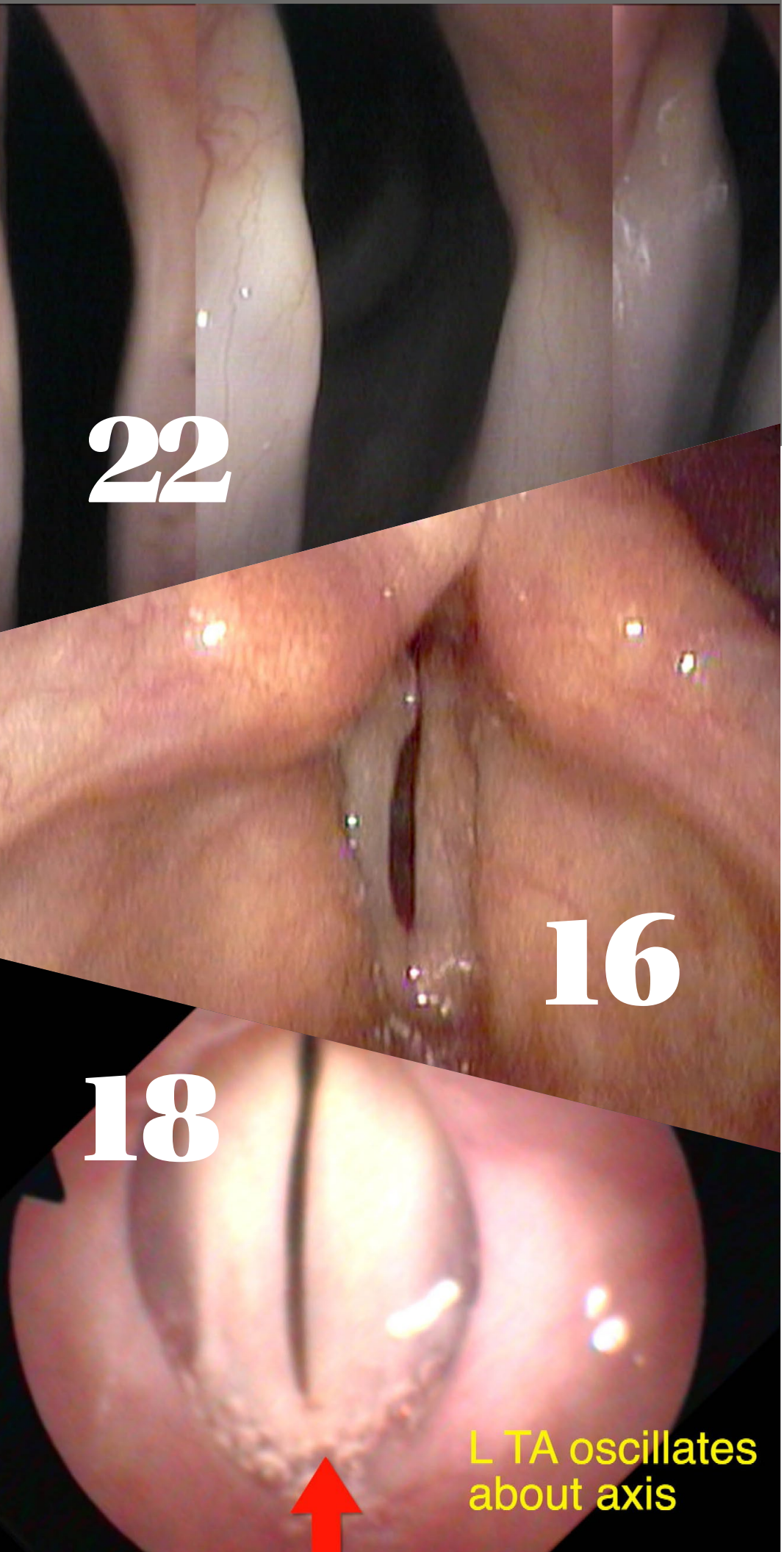
Secrets of Neurolaryngology

LISTEN CAREFULLY
LOOK CLOSELY

JAMES P. THOMAS, M.D.
VOICEDOCTOR.NET/NEURO





SUMMARY

James P. Thomas, MD
909 NW 18th Avenue
Portland, OR 97209 - USA

voicedoctor.net
thomas@voicedoctor.net
+1 503 341-2555

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Neurolaryngology

Becoming a neurologist of the larynx takes time to observe and reason about the functions of each muscle of the larynx. There are three muscles on each side of the laynx innervated by the recurrent laryngeal nerve and one muscle on each side innervated by the superior laryngeal nerve and one essentially midline muscle, so we could say there are 9 muscles of the larynx. If there is only a binary on and off possibility for each muscle duing function, then in theory we have a possibility of 9^2 possible configurations of the larynx or 81 different positions that the vocal cords could assume. Of course in practice there are less than this because the muscles usually work in harmony on either side of the larynx.

However, after an injury, a muscle might just be weak and function partially, so now we have at least three possible configurations for each muscle. No innervation, partial innervation, complete innervation, so we could say 9^3 possible visual configurations of the larynx or 729 different configurations.

The nerve supply to the larynx is very robust. After a nerve injury, nerves will typically regrow. In this process of regrowth, the wrong nerve could supply input to a muscle. The nerve for the PCA might go to the TA muscle leading to contraction at an inappropriate time. Now each muscle could have at least 4 states: no function, normal function, partial function, function at an inappropriate time. So now we have at least 9^4 or 6541 different possible configurations. This is certainly potentially very confusing.

To simplify, the examiner benefits from a video recording over several cycles of a function, including respiration and then phonation. Reviewing the film slowly and repetitively, looking at one function repeatedly and comparing side to side a pattern will usually be discernable. The same film can be reviewed again looking at a different function or area of anatomy.

Reviewing motion of different portions of the larynx separately will lead to an understanding of when the muscles, and hence nerves, are firing. You will have a visual understanding of the neurophysiology of the larynx. Here are a few secrets to winnowing the complexity of movement down to a specific diagnosis.

Secrets of a neurolaryngeal examination

Observation

allow movement to happen repetitively.
isolate laryngeal functions.

FUNCTIONS
- respiration
- phonation

RESPIRATION
- inspiration
- expiration

PHONATION
- high pitch
- low pitch

then

- high pressure/flow/volume
- low pressure/flow/volume

Visual States

STABLE
- paralysis
- paresis
- crossed reinnervation
  - synkinesis
  - dyskinesis

UNSTABLE
- tremor
- spasm

MIMIC
- fixation

Techniques

GET CLOSE
- topical anesthesia
- doesn't impair movement

OBSERVE
- patience
- sniff to stretch
- lift arytenoids
- scope between false cords
- two perspectives

Visual diagnosis of laryngeal neurologic impairments is not only possible, but is the most accurate method for evaluating the neurologic status of the upper airway.

findings
- Atrophy - TA, PCA, False cord
- Fasciculation - TA body, PCA body, LCA arytenoid
- Motion
  - Range of rotation - LCA, PCA (joint, scar)
  - Lengthening - CT (SLN, joint, scar)
- Oscillation - TA, CT
- Hyperactivation - LCA

Tools
- Video endoscope
- Digital recording
- Topical anesthesia
- Close exam
- Vocal manipulation

EMG

Although EMG (electromyography) is commonly spoken of as the "gold standard" (if there even is one), visual examination offers a great deal - let's say a platinum standard.

Assumptions

Muscle action

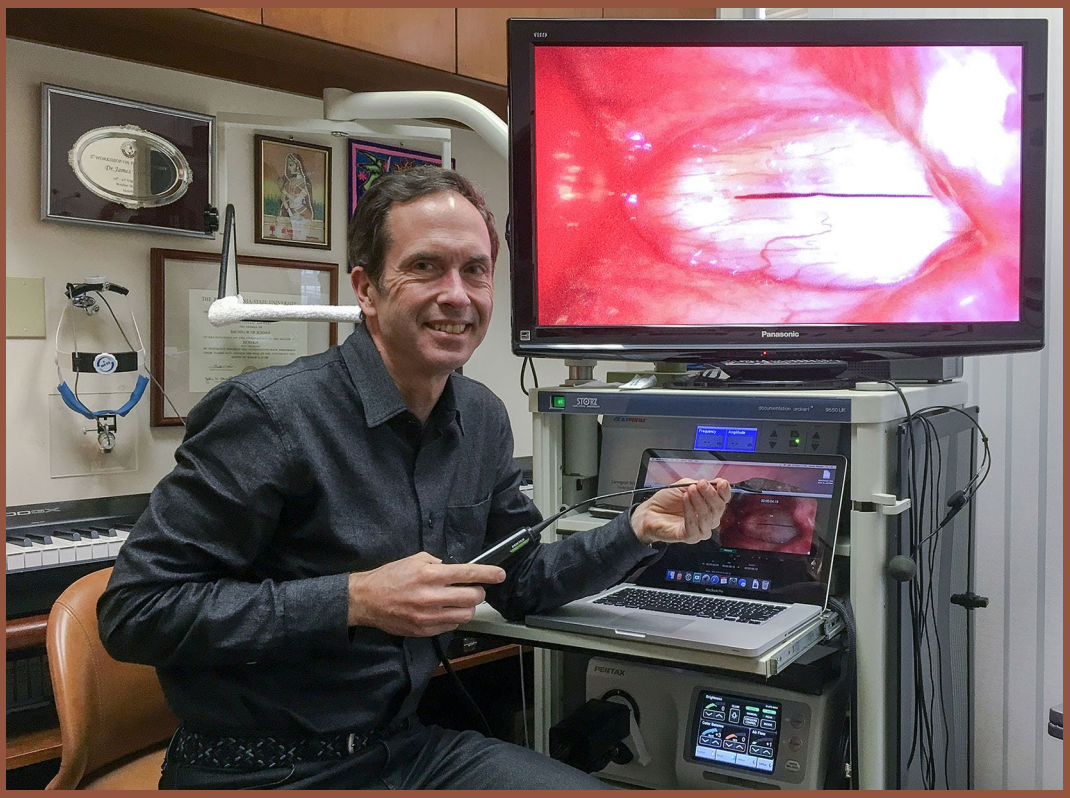
- each muscle has only a single action,
- that action can be isolated,
- that action can be elicited and visualized.

Timing

- each muscle has an appropriate time to contract,
- timing can be compared to the opposite side,
- timing can be compared to expected function,
- inappropriate timing represents reinnervation by an opposing nerve branch.

Compensation

- the patient always attempts to compensate,
- removing compensation reveals pathology.



VISUALIZING

Neurolaryngology: VISUAL EXAM

Careful listening
&
close observation
are more precise
neurolaryngeal diagnostic methods than
laryngeal EMG.

INTRODUCTION

The branching tree structure of the nerve

The first step in visual neurolaryngology is for the examiner to have a mental map of laryngeal anatomy. Understanding the treelike branching of the laryngeal nerves will allow us to work both forwards and backwards from clinical information we have. If we know where and how an injury took place along the path of the nerve, then we will know the findings and impairments that we should see on an endoscopic examination. Likewise we can work in the reverse direction. Based on the functional impairments that are seen on an endoscopic examination, we should be able to predict where along the path of the nerve the injury took or is taking place.

The larynx is supplied by the Xth cranial nerve, the vagus nerve. There are two important anatomic locations along the path of this nerve for the examiner to think about. The first location is where the 10th cranial nerve leaves the skull and heads towards the neck. It passes through a very narrow opening along with two other cranial nerves, the XIth and the XIIth cranial nerves. From a diagnostic perspective, this bottleneck then is a location where an injury to the nerve will likely affect not only the Xth cranial nerve but also the XIth and the XIIth cranial nerves. So in our endoscopic upper airway examination, if there are problems with the tongue (XIIth cranial nerve) or with lifting the arm and shoulder up (XIth cranial nerve), in addition to problems in the larynx, we will know to focus our attention on this narrow opening at the base of the skull.

The second diagnostically important location is where the Xth cranial nerve descends in the neck, it gives off four branches to supply the throat and larynx. Knowing which of these branches are affected, will tell us how high the location of the injury is in the neck. If only single branch of the nerve is involved, we know the injury is taking place very near to the larynx. If multiple branches are involved, we know the injury is taking place further away from the larynx and closer to the brain.

The extended path of the left recurrent laryngeal branch of the Xth cranial nerve descending into the chest and wrapping around the aorta and left bronchus while the right typically remains recurrent in the lower neck is one of the most well known diagnostic asymmetries.

Since speech and voice overlap in function, assessment of the tongue, soft palate muscles and pharyngeal constrictors contribute to understanding laryngeal neurologic injury.

INTRODUCTION

Regrowth

A second principle of neurolaryngology is that the Xth cranial nerve, when injured, almost always tends to regrow and supply muscles with nerve input. Mild injuries and injuries close to an individual branch of the nerve will tend to recover with normal function.

More severe injuries and more proximal injuries, that is, injuries that are closer to the main trunk of the nerve, will result in regrowth with crossing of nerve pathways to different branches. This will result in various degrees of mixed up functioning of the muscles of the larynx. Even in cases where there is a gap between the cut ends of the nerve, the cut neurons will often gradually grow through the intervening tissue and reconnect with the denervated muscles.

BILATERAL SYMMETRY

In many injuries of the larynx, we have a built in reference point. When one laryngeal nerve is injured, the other side continues to function normally and provides the examiner with a direct comparison by way of asymmetry in anatomy and function between the two sides. We can compare the structure on the left with the structure on the right. We can also compare the timing and range of movement on one side with the other.

Muscles

Some of the muscles innervated by the laryngeal nerves are so close to the surface mucosa that they can be nearly directly visualized. Others are deeper and only their effects can be visualized. Near direct visualization includes the ability to see the muscle contract and thicken or shorten. Atrophy and fasciculations can be visualized beneath the mucosa.

Indirect visualization is the ability to infer muscle contraction from joint and structure movement.

Some activities, such as respiration, are rhythmic and symmetric. Inappropriate timing and asymmetric motion can be utilized to infer innervation status.

Muscles observed during a neuroendoscopic evaluation:

- lingual muscles - genioglossus
- palatal muscles - levator veli palatini
- pharyngeal constrictors
- laryngeal muscles
  - thyroarytenoid (TA)
  - lateral cricoarytenoid (LCA)
  - posterior cricoarytenoid (PCA)
  - interarytenoid (IA)
  - cricothyroid (CT)

Near direct visual findings include the following (along with the muscles in which they can be endoscopically observed):

Atrophy - TA, PCA
Fasciculation - TA, PCA

Indirect visual findings utilized to infer muscle function include the following (along with the muscles in which they can be endoscopically observed):

Oscillation - TA, CT
Tension - TA, CT
Range of motion - LCA, PCA
Lengthening - CT
Respiration
- Inspiration - PCA
- Expiration - LCA

Timing - inappropriate reinnervation
Compensation - unmasking

Findings

- Atrophy - TA, PCA, False cord
- Fasciculation - TA body, PCA body, LCA arytenoid
- Motion
  - Range of rotation - LCA, PCA (joint, scar)
  - Lengthening - CT (SLN, joint, scar)
- Oscillation - TA, CT
- Hyperactivation - LCA

Motion - Movement
slow & fast

Contraction --> movement

SLOW

MUSCLE	EFFECT	ACTION	OBSERVE
TRANSLATION			
PCA	**ABduction**	Opening glottis	vocal process / posterior larynx
LCA (& TA - IA)	**ADduction**	Closing glottis	vocal process
CT	**tension (extrinsic)**	Elongate vocal cord	vocal process / anterior comm.

FAST

OSCILLATION			
TA	**tension (intrinsic)**	oscillation rate	vocal margin amplitude
CT	**tension (extrinsic)**	oscillation rate	vocal margin amplitude
FLUTTER			
no muscle	flaccidity	flutter	membranous margin

Larynx motion appears to be complex, at least on superficial examination. Some of the apparent complexity in reference articles derives from failure to discriminate among the various types of motion. I frequently read the statement, "The vocal cords don't move." I don't know whether the speaker is referring to the translational movement of abduction and adduction or oscillation impairment (perhaps from intrinsic stiffness of the vocal cord). They usually are not referring to lengthening of the vocal cord, which is also a movement.

When I read a report that says, "The vocal cords don't move," I also suspect that there is no distinction being made between a lack of abduction / adduction from:

- a lack of muscle contraction vs.
- inappropriate or simultaneous attempted abduction and adduction leading effectively to no or subtle translational movement.

Further:

- This subtle movement may be reduced but:
- appropriate or inappropriate in direction,
- appropriate or inappropriate in timing.

Impairment

Paralysis

Complete loss of voluntary movement

Paresis

Partial loss of voluntary movement. Normal timing and direction of motion

Crossed reinnervation

Synkinesis

Simultaneous stimulation of two muscles that cancels movement

Dyskinesis

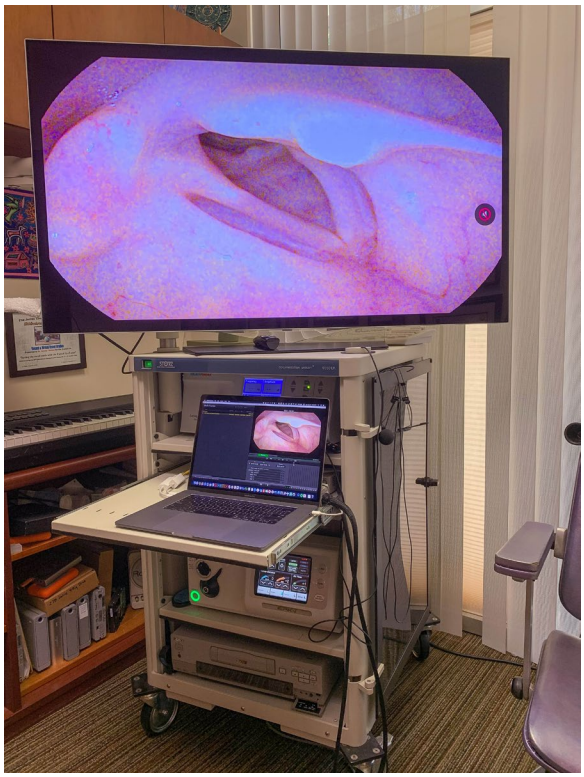
Stimulation of a muscle that leads to inappropriate (timing & degree) of movement

Paralysis may be the most frequently used term, yet it is the rarest condition. Shortly after a complete transection of the nerve is almost the only time I can be confident of a complete paralysis.

Paresis is a relatively common condition in which a muscle fails to completely contract. There is partial and appropriate motion.

Given the high incidence of nerve regrowth, synkinesis and dyskinesis are frequent findings.

Tools



Tools

- Video endoscope
- Digital recording
- Topical anesthesia
- Close exam
- Vocal manipulation

VIDEO ENDOSCOPE

The most common tools of the laryngologist; endoscope, processor, stroboscope are likely in almost every office. So too is some recording device, though too often the audio portion of the recording seems to be left out.

TOPICAL ANESTHESIA

Topical anesthesia is essential in some cases to see beneath the arytenoids or between completely compressed false vocal cords. Topical anesthesia with lidocaine does not alter vocal cord muscle motion. It only eliminates sensation temporarily, typically long enough to perform a detailed, close-up examination.

DIGITAL RECORDING

A digital recording device may also be common although many computer configurations are more difficult to use than a typical laptop with standard video software. The ability to look at a video clip,

- repetitively,
- in varying degrees of slow motion,
- in forward and reverse, and
- frame-by-frame are excellent software tools for the neurolaryngologist.

CLOSE EXAM

Close examination can sometimes be performed during quiet respiration. However, true close examinations during phonation often require topical anesthesia.

Topical lidocaine, applied to the vocal cords allows the endoscope to lift the arytenoids out of the way and view the vocal processes in nearly all situations. The vocal process is the key guide to LCA and PCA muscles.

Topical lidocaine allows obstructing false cords to be moved laterally and true cord oscillation visualized directly in situtations of vocal weakness.

VOCAL MANIPULATION

It is easy to forget vocal manipulation as a tool. Eliciting high and low pitch as well as high and low air flow assesses the function of the laryngeal muscles in different configurations of neural stimulation. Changes in position of the vocal cords and in the pattern of oscillation will reveal weakness.

Observation

Take time

Observation

take time to stop and allow movement to happen repetitively

FUNCTIONS

- respiration
- phonation

RESPIRATION

- inspiration
- expiration

PHONATION

- high pitch
- low pitch

then

- high pressure/flow/volume
- low pressure/flow/volume

It is easy to go for the gold. Spray the nose, put the endoscope in and look at the vocal cords for a lesion. Often the examiner's emphasis is on structure. However, laryngology is about function.

Observing function requires video more than photos. Value lies in observing the movement of structures over time.

Although neurolaryngology takes time, that time can be used efficiently. During the general portion of the exam, as soon as one sees which nasal passage is more open, spray a mixture of a decongestant and topical anesthetic into that nasal passage. By the time the general exam is finished and patient's vocal capabilities recorded, the nose is well anesthetized. Patients are amazed how comfortable this exam is compared to a quick endoscopy.

Taking time extends to the related cranial nerves. Before looking at the vocal cords, assess the movement of the tongue, soft palate and pharynx.

Next, with the entire larynx in view, record several cycles of respiration. It seems like an eternity while recording, but when reviewing videos later, 5-7 cycles of quiet respiration is quite minimal.

If one hasn't seen the vocal processes on an overview, move the endoscope closer and again record several cycles of respiration from this vantage point.

The same applies to stroboscopy. Recording a single pitch misses too much information. Record a range of pitches. Record a range of volume. Record both loud and soft sound production at low pitch and then loud and soft again at a high pitch. Recording a glide from low to high pitch and a glide from high to low pitch reveals additional information.

Endoscopy exams typically range from about 40 seconds of recording up to 4 minutes for more subtle problems. Stroboscopy exams are also typically 40 seconds up to 3 minutes of recording.

At times, reviewing the same motion multiple times is warranted. Breaking down respiration into components will yield additional clues. For example, focus on inspiration only for several cycles. Then review the same recording of breathing cycles focusing on expiration only. Return and review just the right vocal process movement several times, sorting out range of motion as well as timing and comparing it to the opposite vocal process for range of motion and timing of motion when they are not synchronous. This component review of the video takes additional time, but reveals otherwise missed details.

Xth Cranial nerve
palatal branch
pharyngeal branch

Supra-laryngeal branches

UPPER GANGLION BRANCH (SUPPLIES PALATAL LEVATOR)

ORAL VIEW OF PALATE

- Observe midline pulling lateral during /ah/
- Observe indentation in soft palate during /ah/

NASAL VIEW OF PALATE

- Both sides can be seen from one nasal passage
- Normal elevation is complete without air leak during plosives (/pa/, /ka/, /ta/) and fricatives (/sh/, /th/)
- Abnormal elevation is incomplete
- One side lack of complete elevation - gap - especially if viewed from pathway over the middle turbinate
- Bubbles from secretions during phonation - plosives and fricatives
- Can use endoscope as occlusive test - during whistling - knowing diameter of scope
- Also helps patient to realize the extent of injury - often the loss of voice is most obvious injury and may cover up symptoms of nasal leak which can then later become very noticeable after any laryngeal correction.

LOWER GANGLION BRANCH (SUPPLIES PHARYNGEAL CONSTRICTORS)

Position endoscope in upper pharynx, observe midline during a vocal glide from highest pitch to low

- Observe midline raphe which pulls toward strong side
- Observe lateral pharyngeal squeeze from constrictors
- Coincides with pooling/ open piriform sinus

Superior Laryngeal Nerve
Cricothyroid muscle

SYMPTOMS
- Loss of power
- loss of upper range
- Loss of vocal control during singing (example: loss of easy pitch matching)

HISTORY
- frequently injured during neck surgery (thyroid, parathyroid)

DIFFERENTIAL DIAGNOSIS:
- Bilateral SLN injury is relatively uncommon and symmetric bilateral motion impairment more often represents scar or fixation

FINDINGS
VOCAL CAPABILITIES BATTERY:
- Limitation of the extent of upper notes compared to expected range (or to examiner)
- Flatting and poor control in the upper vocal pitch range

ENDOSCOPY
- Vocal cord length changes minimally, less than expected or not at all during attempted glide up in pitch

STROBOSCOPY:
- Symmetric oscillations at low pitch while
- Abnormal findings at high pitch
  - Asymmetry of vocal fold vibration at high pitch present because differential tension is most evident when only one cricothyroid muscle contracts.
  - At high pitch the injured (non-tense) cord oscillates lateral to it's axis while the normal cord oscillates symmetrically about it's axis
  - At phonatory onset and offset the weak cord has a central glottic gap

Recurrent Laryngeal Nerve
Posterior Cricoarytenoid muscle

SYMPTOMS
- stridor
- none

HISTORY
- injured during surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with change in voice

DIFFERENTIAL DIAGNOSIS:
-

FINDINGS
VOCAL CAPABILITIES BATTERY:
- No vocal abnormalities from this muscle

ENDOSCOPY
- Arytenoid tipped forward
- Posterior laryngeal fasciculations
- Atrophy of PCA area during sniffing
- No lateral motion of arytenoid during sniffing

Usually the more distal branches of the LCA and TA muscles are also affected

OTHER NOTES:
- PCA neurons have a strong tendency to grow back to both the anterior and posterior branches of the RLN
- This usually weights activation of the RLN during respiration toward adduction of the recovered branch

Recurrent Laryngeal Nerve
Lateral Cricoarytenoid muscle

SYMPTOMS
- weak voice (hoarseness)
- delayed: laryngospasms

HISTORY
- weak voice immediately after surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with prolonged loss of voice

DIFFERENTIAL DIAGNOSIS:
- Bowing may mimic some symptoms
- Nonorganic dysphonia
- delayed: dyskinetic reinnervation may be confused with asthma

FINDINGS

VOCAL CAPABILITIES BATTERY:
- loss of lower vocal range (CT aids closure in upper range so less air leak at high pitch)
- delayed: normal

ENDOSCOPY
- focus on vocal proces position and orientation
- watch respiration for reduced range of motion (ROM) between inspiration and expiration
- delayed: vocal process midline, but immobile
- delayed: vocal process crosses midline from PCA innervation

ENDOSCOPY OR STROBOSCOPY
- watch vocal process closure during phonatory onset (adduction) in slowed down motion for speed of closure and range of closure.
  - view multiple closures to differentiate from nonorganic muscle tension where closure will be variable and intermittently complete
- Upper portion of arytenoid will often cover view of vocal process at end of closure, earlier if the PCA is out on the same side. Low speed very valuable.
- topical anesthesia greatly enhances view of posterior commissure during phonation - best view
- Observe for triangle of lateralized vocal process on weak side. Be cognizant of hyperclosure from good cord's vocal process
  - View at varying pitch (high, medium and low pitch)
  - lowest possible pitch reveals largest triangle.
- with increased volume, arytenoid can be pushed laterally at low pitch in weak LCA, which partly corrects with pitch elevation as CT contraction compensates at higher pitch

STROBOSCOPY:
- no obvious oscillatory change unless other muscles are paretic. TA & LCA are frequently paretic, synkinetic or dyskinetic together

OTHER NOTES:
- View of rigid exam with small "apparently symmetric" posterior gap that changes to an asymmetric gap when viewed up close on flexible exam.

Recurrent Laryngeal Nerve
Thyroarytenoid muscle

SYMPTOMS
- weak voice (hoarseness)
- delayed: reduced vocal range

HISTORY
- weak voice immediately after surgery (thyroid, parathyroid, carotid, esophagus, left chest surgery)
- cold symptoms with prolonged loss of voice

DIFFERENTIAL DIAGNOSIS:
- Bowing may mimic some symptoms

FINDINGS
VOCAL CAPABILITIES BATTERY:
- loss of lower vocal range (CT provides some tension for upper range so less air leak at high pitch)
  - obligate falsetto - high comfortable speaking pitch
- husky noise predominantly in lower range
- diplophonia in lower range from asymmetric true cord mass, asymmetric true cord tension
- delayed: normal

ENDOSCOPY
- focus on membranous true vocal cord
- bowing - rigid scope
- atrophy - flexible scope best for visualization
  - sometimes requires topical anesthesia to view vocal cord thickness or mass
  - Sniffing stretches the vocal cords and augments subtle atrophy.
    - Slow motion helpful for viewing thinning secondary to sniffing
    - Phonation first, followed by sniffing also augments thinness.
  - Can infer atrophy from enlarged ventricle
  - Inspiratory bernoulli effect if atrophic and if near opposite vocal cord
- fasciculations (visible on superior vocal cord surface as well as in subglottis)
- lack of tension

STROBOSCOPY:
- TA weakness is augmented at lower pitch
  - Low pitch removes cricothyroid compensation in partial paresis
  - If supraglottic squeeze is predominant only at low pitch - implies glottic incompetence more often than intentional hyperfunction
    - Topical anesthesia allows passage of endoscope between false cords for a view of cords during phonation
- Oscillation
  - Low pitch
    - oscillates lateral to axis
    - Excursion amplified at low pitch
  - High pitch
    - CT keeps cords near each other
    - CT keeps cords in phase or almost in phase (patients tend to avoid diplophonia - especially during an exam)
- Augment abnormal vibrations by increasing subglottic pressure (volume and airflow). Visualized as
  - Flutter - random oscillation, or
  - Biphasic - one central node of oscillation with two segments

REINNERVATION

NORMAL RECOVERY

- Occurs after a minor injury
- Restoration of complete range of motion
- Restoration of appropriate intentional motion

SYNKINETIC RECOVERY

- Occurs after a moderate injury
- PCA neurons split reinnervation to anterior adductor muscles and posterior abductor muscles
  - On many occasions the vocal cords on endoscopy appear immobile, resting in or lateral to the midline,
  - or move slightly and movement may be appropriate in direction.
  - Tension is maintained during phontion, rather than flutter.
  - Many times the reinnervated vocal cord is tighter than the "normal" cord during phonation in the vocal underdoer or aged person with bowing of the "normal" cord.
  - Perhaps this represents a nearly 50:50 arrangement when abductor and adductor neurons reinnervating LCA and TA.
  - There may also be a mix of abductor and adductor neurons reinnervating the PCA .
  - If normal recovery doesn't recur, this is the next best "ideal" spontaneous recovery.
  - That is, it does not require intervention by surgeon.

DYSKINETIC RECOVERY

- More severe injury
- Overgrowth of PCA (abductor) neurons to LCA muscle
  - Vocal process may cross the midline
  - Vocal process may be highly angled toward midline
  - Vocal process may move in the opposite direction of intention
    - medial during inspiration
    - lateral during phonation
    - lateral during expiration
- Dyspnea or stridor
  - May be light to very strong
  - Not due to weakness, it is hyper reinnervation
- May occur 10 – 20 years after injury
  - Often misdiagnosed as unresponsive asthma because of remote interval from injury
- Dyskinetic activity may increase with vocal use
- High rate of spontaneous laryngospasm
  - Responds to botulinum toxin injection into "paretic" cord.
  - Responds to reinnervation of anterior branch with ansa cervicalis with improved stability and steady tension during phonation

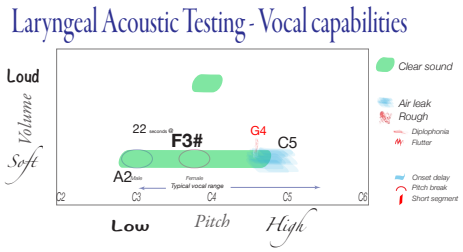
MAIN ISSUE IN BILATERAL RLN INJURY

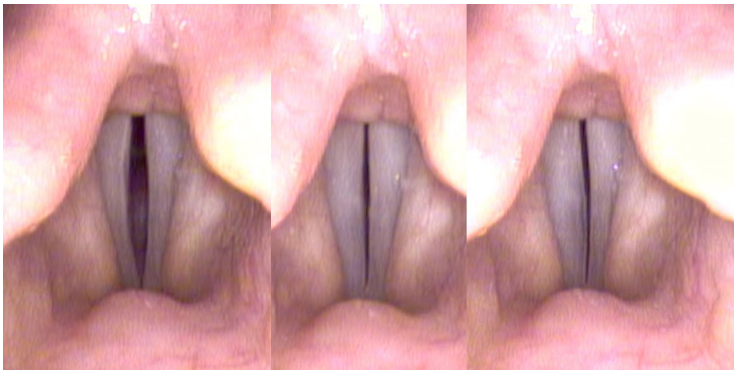
- Initial symptoms after bilateral injury
  - Weak voice
  - Partially out of breath
  - Choking on water
- Symptoms after several months
  - PCA (abductor) neurons activate LCA movement toward midline
  - Louder voice, more dyspnea
  - More stridor
  - Increase frequency and intensity of laryngospasm
- Patients often learn to relax TA muscle and/or CTA muscle during inspiration
  - Shortens vocal cord
  - Allows central membranous cord to bow laterally
- Botulinum toxin injection
  - If placed into LCA muscle, tends to improve airway
  - If placed in TA muscle,
    - may precipitate functional airway obstruction
    - bernoulli effect moves membranous cord to midline during inspiration

Infering tension from oscillation

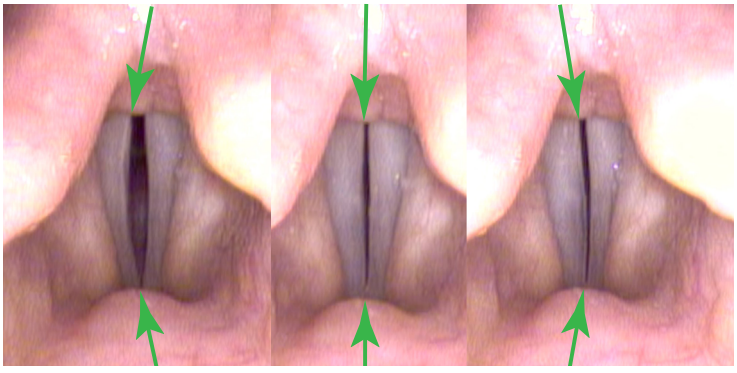
Stroboscopy

Two years ago Sally (50's) began losing her upper singing range. Her singing endurance dropped to where she fatigues after 15 minutes. She is even having difficult singing alto now.

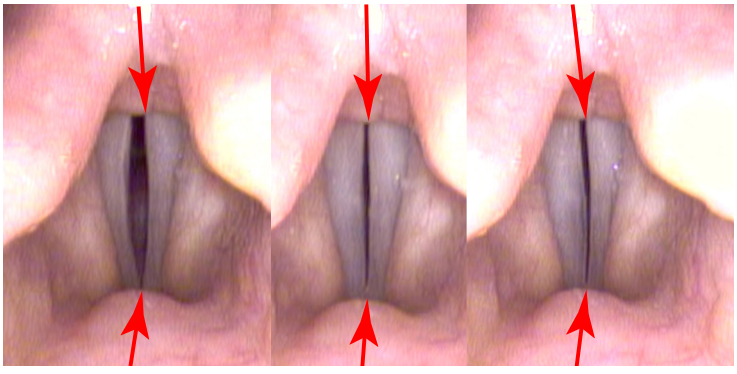




Three selected frames from the stroboscopy exam at pitch G4 where her voice is straining to reach the note and she is leaking air.



If we follow the oscillation pattern of the right vocal cord (green arrows), the right vocal cord's central margin oscillates from lateral (left frame), to midline (middle frame) then crosses its axis (right frame) moving the vibratory margin medial to its axis. This cycle repeates over and over. The right cord is oscillating nearly symmetrically about its axis – because of adequate vocal cord tension.



If we follow the oscillation pattern of the left vocal cord (red arrows), the left vocal cord starts lateral (left frame), moves almost to midline but has a slight concavity (middle frame) and then return to a lateral to its axis position (right frame). This cycle repeats over and over.

The left vocal cord has insufficient tension relative to the right vocal cord and at this pitch (G4) never crosses its axis.

When she was examined at lower pitches, the vocal cords were symmetric in their oscillations, meeting in the midline.

In her lower range, when the cricothyroid is not activated, the tension in each vocal cord is the same leading to symmetric oscillations. As she increases pitch, the tension is greater on the right vocal cord (intact cricothyoid muscle).

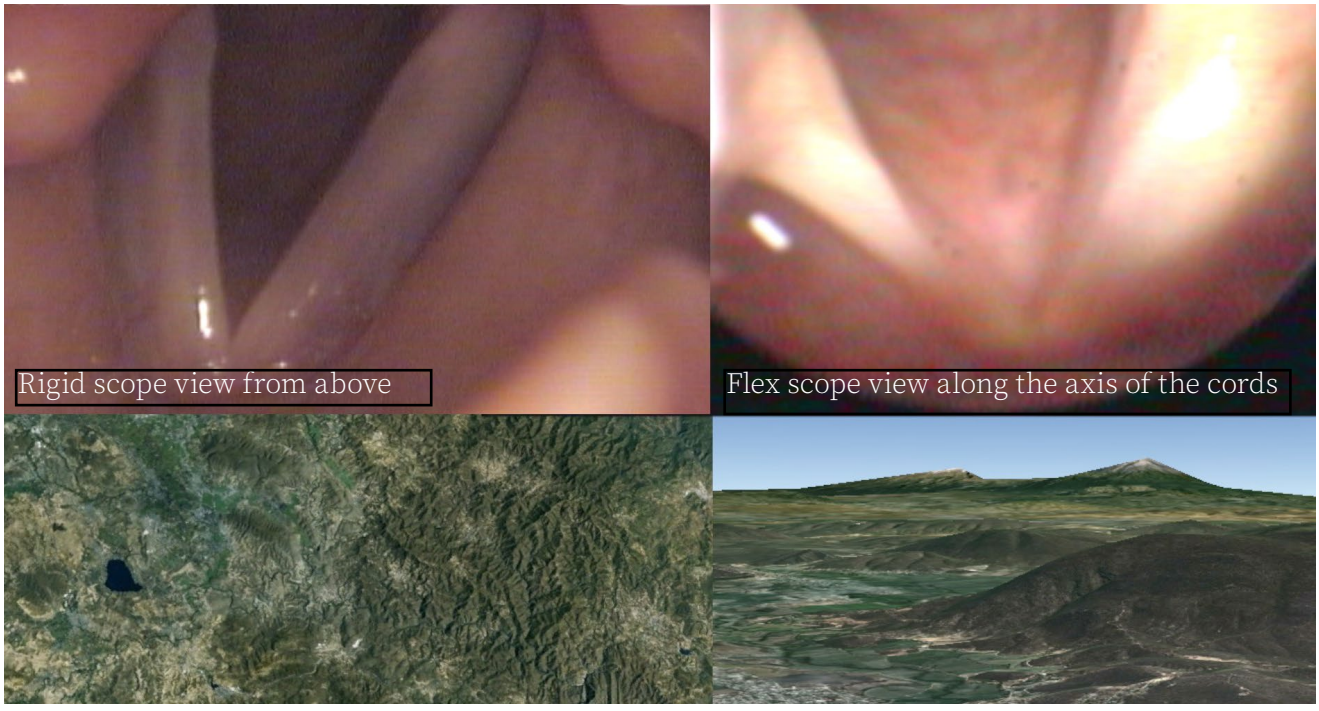
This case represents a left superior laryngeal nerve paresis.

Using two endoscopes for perspective
Get super close

Position vs. Mass

Two endoscopes can offer different perspectives. The rigid endoscope view is high quality but usually more limited in terms of perspective.

A flexible endoscope can be passed into the laryngeal introitus and curved to approximate the axis of the true vocal cord giving a nearly parallel view. Often topical anesthesia is required to obtain this super close up view.



Satellite type view on a map
(Map Data: Google, © Cnes/Spot Image 2013)

Airplane type view: same region on a map
(Map Data: Google, © Cnes/Spot Image 2013)

Vocal cord position can explain some symptoms such as a weak or breathy voice when the vocal cords cannot approximate. But what if there is complete closure of the glottis from the moveable vocal cord approximating the immobile but midline vocal cord, yet the patient has an obviously diplophonic voice. A vocal cord resting in the midline does not explain diplophonia. However, the "airplane view" of the flexible endoscope reveals the difference in mass between the vocal cords and two vibratory structures of differing mass will tend to vibrate at different frequencies, consequently explaining diplophonia.

Dyskinesia & paresis
Paralysis – paresis – synkinesis

Dyspnea

Catherine had her thyroid gland removed 30 years ago. She developed a hoarse voice after the surgery suggesting her recurrent laryngeal nerve had been injured. Stretched, cut or somehow traumatized, the nerve stopped working after the surgery and one of her vocal cords could not move close enough to the other vocal cord to start vibrating with air flow, so she had no voice for several months.

Gradually her voice returned and eventually it recovered – or more likely it seemed to recover. She could no longer sing nor reach high pitches, but day-to-day, her voice was adequate for work. She accepted that as recovery.

Gradually over the past several years she started having sudden episodes of her breathing being cut off. She would be speaking and suddenly she couldn't breathe.

Every otolaryngologist who looked told her she had a paralyzed vocal cord, but that she still had "plenty of room to breathe." Several physicians told her that her episodes of difficulty breathing were from silent acid reflux and they placed her on antacids (didn't help).

We should ask, do her brief, sudden, "shortness of breath" episodes (dyspnea, usually with stridor) have anything to do with her previous surgical injury? Phrased another way, are laryngospasms related to nerve injuries?

The word paralysis means lack of mobility. It is true that her injured vocal cord does not obviously open or close with each attempted phonation or each attempted breath. There is the implicit assumption that since her vocal cord is not moving normally there is a lack of innervation – an error. The recurrent laryngeal nerve actually has such a strong propensity to regrow that even after excising several centimeters of the nerve, it still often grows back to the muscles in the larynx. When the nerve grows back, the major issue is not lack of nerve input, but lack of proper nerve input.

We can say that she is suffering from a laryngeal dyskinesia. Calling her injury a laryngeal dyskinesia implies different findings and different problems than laryngeal paralysis. A dyskinesia may be present whether or not there is any observed motion impairment, though usually there is some impairment of motion. After a nerve injury the problems that result are, to some degree, due to the degree of reinnervation, but even more due to inappropriately directed reinnervation.

In the ideal world, the injured nerve would regrow back to the muscle it used to control. In the most typical severe nerve injury about half the fibers end up going to their original muscle and the other half go to the opposing muscle. Consequently, the neurologically injured vocal cord appears to be immobile. The brain tells both muscles to contract simultaneously and the net effect is that there is no motion and nothing obvious happens.

That seemed to be the case with Catherine for many years, but something definitely changed in recent years. She began having the laryngospasms that cut off her breathing entirely for a seeming eternity (when in reality it was less than a minute, but when you can't get air, time subjectively moves slower).

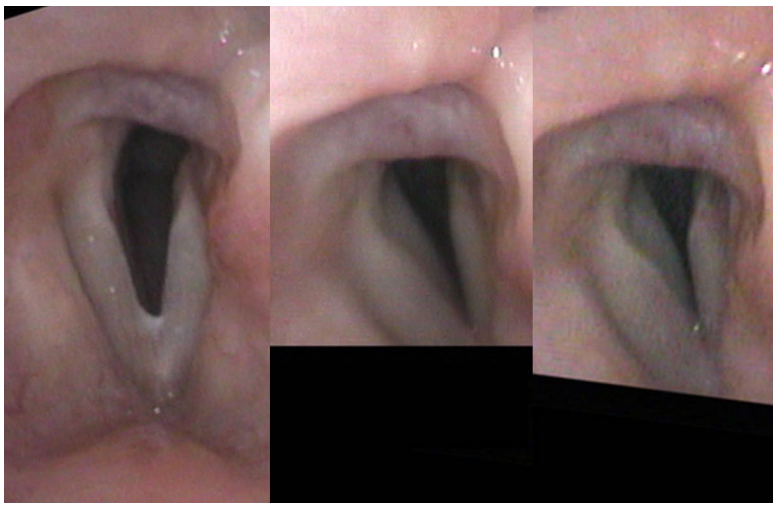
On her endoscopic exam, the healthy right vocal cord opened and closed appropriately, both during breathing and during sound production. She had quite a strong voice, though I would say that it actually had a strained or tight quality.

During the ultra-close portion of my endoscopic exam, I touched the left, non-moving or paralyzed cord lightly and it suddenly moved across the midline nearly closing off her airway. It wasn't paralyzed; it could move. It just did not move intentionally and appropriately during breathing or during phonation and it was trigger-happy. With even a small trigger, her left LCA muscle would spasm and move the left vocal process nearly all the way to the opposite cord.

This LCA spasm also increased gradually the longer Catherine spoke. With each phonation the right cord would touch the left side and the left vocal process would move further toward the right after each touch. Then, after resting her voice for a number of breaths, the left side would relax back toward its midline, resting position. (see photos below)

As both treatment and a test, I injected botulinum toxin into her dyskinetic (actually the opposite of paralyzed – hypercontracting), left vocal cord. Specifically, I placed the botulinum toxin into the TA and LCA muscles – the muscles that tense and move the cord toward the midline and closure.

Two weeks later, the opening in her larynx was larger while she was breathing. She could



Left: vocal cords at initial rest during inspiration.
Middle: after the stimulation of phonating, the left vocal process starts moving across the midline during inspiration. It remains in this position even during expiration.
Right: left vocal process hyperadducting during inspiration and narrowing the airway further after more vocal stimulation.

still make sound, though a little more softly. However, she could breathe better than she had in several years and she had not had any further laryngospasms since the injection. The paralytic effect of botulinum toxin lasted for three to four months and then the nerve connected to the muscles again and the left-side began to hypercontract again, which she could identify because of the increasing difficulty with breathing. Catherine returned to the office for another treatment every few months. After several injections, she asked if there was something more permanent that could be done.

The RLN splits like a tree into different branches. It is possible to cut only the branches that go to the closing muscles (the TA and LCA) and in effect, that is what we were doing chemically with the botulinum toxin. I suggested a surgery where we would cut the anterior branch of the RLN. Then, to prevent the original RLN from growing back as it had done 30 years ago after her injury, I would route a nerve from one of her neck muscles (the omohyoid) into the cut anterior branch suppling the TA and LCA muscles. If this new nerve sprouted fibers to the muscles before the old branch of the RLN, then she would have nerve input to these muscles during phonation (the omohyoid tenses during phonation). The TA and LCA muscles would bulk up, and even if the muscles didn't have a completely appropriate signal to move open and closed, they would hold tension during phonation and would not tend to inadvertently spasm nor tighten during breathing in.

After cutting the anterior branch of the recurrent nerve during surgery and sewing in the donor nerve, she had a weak voice for a month and then her vocal strength began to return. The left vocal fold ultimately positioned itself near the midline leaving a much larger opening than when I first met her and she could breathe easily (photo below). She no longer had laryngospasms that would cut her breathing off and her voice was less tight and strained. She could close the right vocal cord all the way to the left and had a strong and clear voice without the tight quality she had previously.

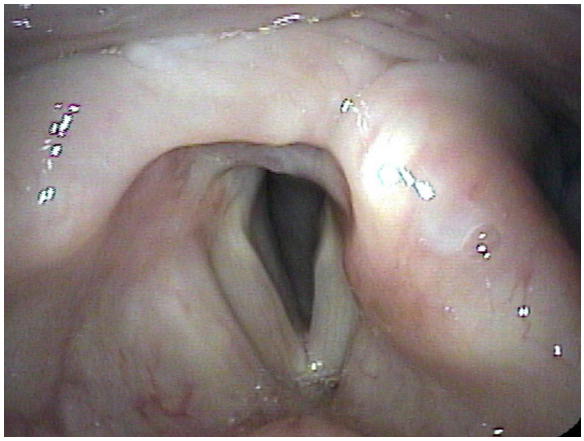
She has a reasonably open airway with the left vocal cord now stable and set in the midline during both inspiration and phonation and very importantly, no further laryngospasms.

A vocal cord's muscles after a nerve injury are seldom really paralyzed, even if there is no obvious easily recognized vocal process movement. The vocal cord doesn't lack movement though it may lack easily visible, intentional movement because of competing contractions. Also, it often has subtle, significant, inappropriate movement.

SUMMARY

After a nerve injury the vocal fold is more likely to move poorly and inappropriately (dyskinesia) than not move at all (paralysis).

For physicians who approach hoarseness after a surgical RLN injury with benign neglect, feeling that a hoarseness that recovers is inconsequential, they might wish to have their patients examined by a laryngologist to better understand their complication rate. A return to a normal voice might not mean a return to normal function.



Laryngeal opening 6 months after reinnervation on left from ansa cervicalis to anterior branch of the recurrent laryngeal nerve the left vocal process remains near the midline during respiration.

EXAMPLE CASE

Lateral cricoarytenoid paresis

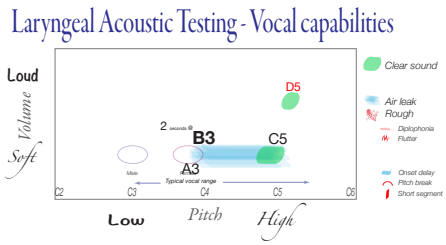
Weak voice - out of breath talking

At age 74, Stella was sitting in the hospital holding her grandchild and lost her voice. She thought it might be from the air conditioning. 6 months earlier she had completed radiation therapy to her chest for breast cancer. She cannot sing anymore and she is out of breath with talking. The first otolaryngologist told her nothing was wrong and that she had forgotten how to talk. She saw a second otolaryngologist about 8 times, trying lansoprazole and famotidine at various doses without improvement.

EXAM

VOCAL CAPABILITIES

Vocal capabilities testing was suggestive of a weakness although a nonorganic voice disorder could be considered in the differential diagnosis.

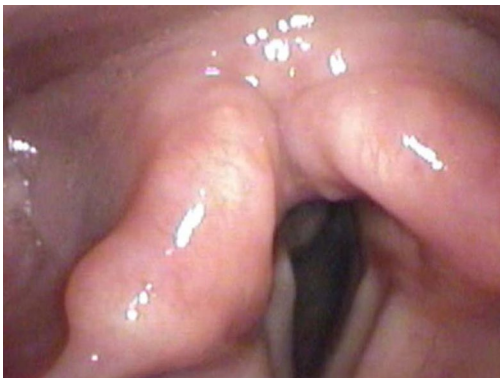


On vocal capabilities exam, she spoke with a falsetto quality. Her maximum phonation time was 2 seconds and she had a breathy quality throughout her vocal range except at high pitch, high volume where her sound was clear.

ENDOSCOPY

On endoscopy, the vocal cord architecture was normal.

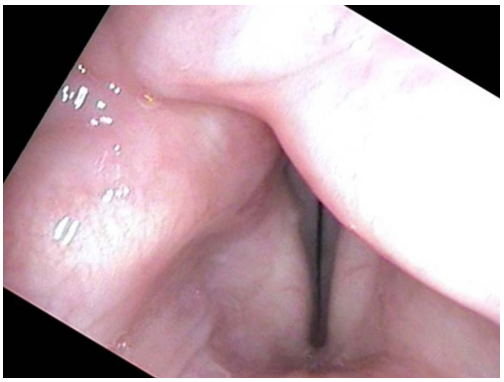
Quiet respiration was observed.



The vocal processes were symmetric during inspiration. However, during expiration (above), the left vocal process did not move as far medial, nor rotate as medial as the right vocal process.

STROBOSCOPY

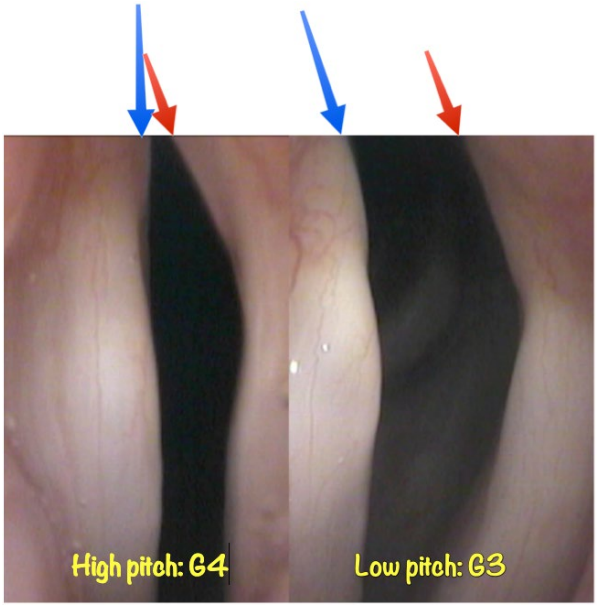
Stroboscopy was performed at her choice of pitch.



When allowed to phonate at any pitch (here vocal cords at phonatory offset at G4), her vocal cords align seemingly perfectly.

While her vocal capabilites supported her complaints, her visual exam didn't reveal an answer. Consequently, her larynx was topically anesthetized and the endoscope inserted beneath the vocal processes. She was then examined at varying pitches.

The tip of the vocal processes hold the key to assessing the functional status of the lateral cricoarytenoid muscle.



At high pitch the right vocal process is near the midline (blue arrow). The left vocal process has not rotated medially (red arrow), but the cricothyroid has pulled both vocal cords longer and thus straightened them, indirectly pulling the vocal processes closer together.

At low pitch the right vocal process is angled past the midline (blue arrow). The left vocal process has not rotated medially (red arrow) leaving a large gap for air escape. This gap is not visible unless the endoscope is placed beneath the arytenoids

CONCLUSION

She has an isolated left lateral cricoarytenoid muscle paresis. Her natural tendency is to use the cricothyroid muscle for compensation.

Thyroarytenoid & Lateral cricoarytenoid paresis

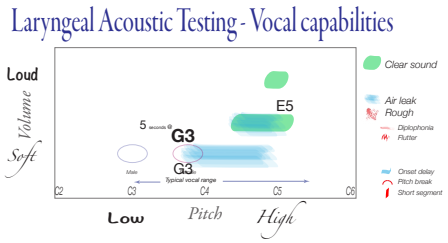
Weak voice - out of breath talking

At age 51, Melanie began to develop a raspy voice 4 days after hip surgery. She was told that she did not have an endotracheal tube in. Her allergist told her it was from reflux and treated her with omeprazole. Her heartburn went away and her voice remained poor. She saw an otolaryngologist who saw a vocal nodule and sent her to Speech therapy. She did not get any better.

EXAM

VOCAL CAPABILITIES

Vocal capabilities testing was suggestive of a weakness, most significantly in her lower vocal range.

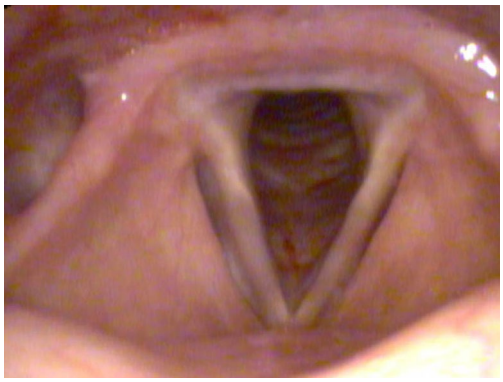


On vocal capabilities exam, she spoke with a lot of huskiness. Her maximum phonation time was 5 seconds and she had a breathy quality throughout her soft and low vocal range. At high pitch, high volume her sound was clearer.

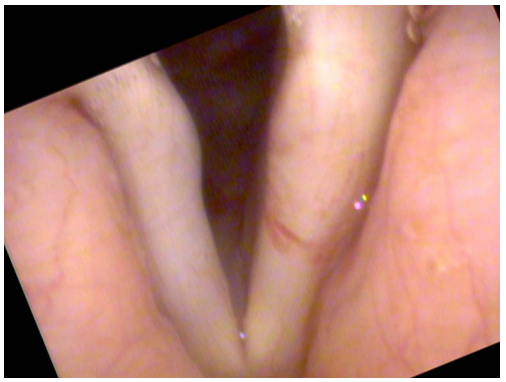
ENDOSCOPY

On endoscopy, the vocal cord architecture was normal on a typical overview.

A close view during expiration also appears almost normal.



Inspiration

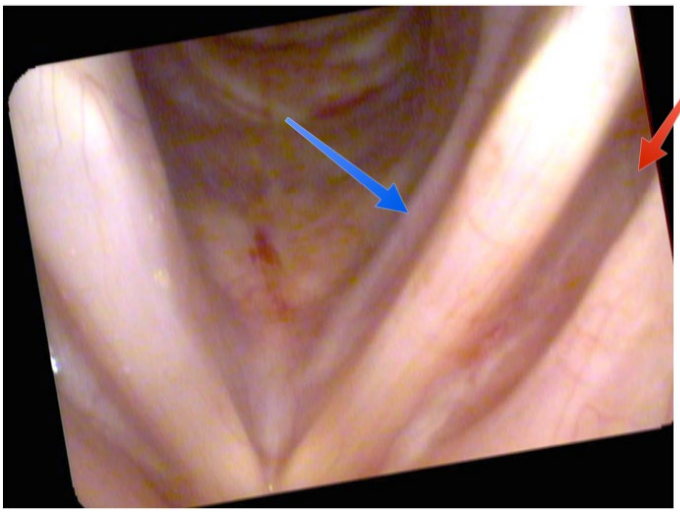


Expiration

Deep inspiration stretches a thin thyroarytenoid muscle.

CONCLUSION

She has an left thyroarytenoid muscle and additional findings show it combined with a left lateral cricoarytenoid muscle paresis. Her natural tendency was to use supraglottic compression for compensation.



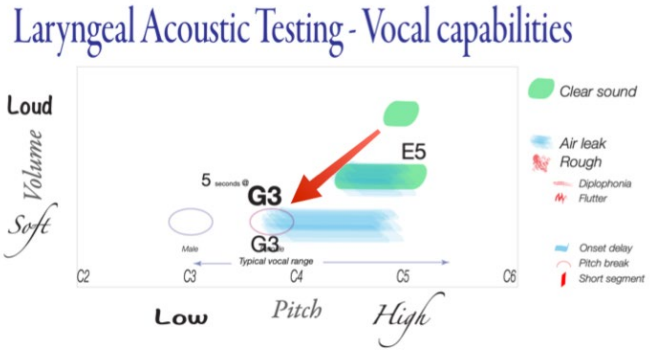
Close up view of deep inspiration reveals concavity of left subglottis (blue arrow) and a large left ventricle (red arrow). There is less mass within the left thyroarytenoid muscle.

VOCAL CAPABILITES TESTING

Neuro- paresis

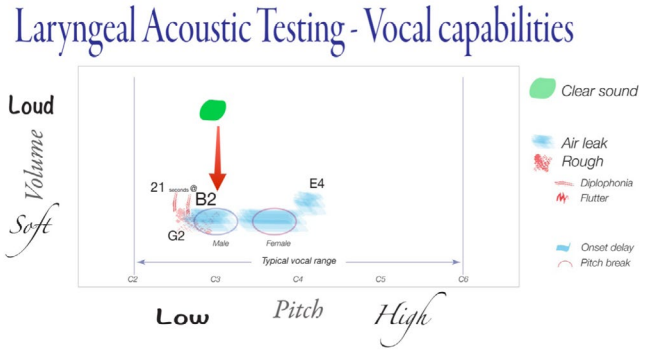
Nonparetic

Paresis TA, LCA



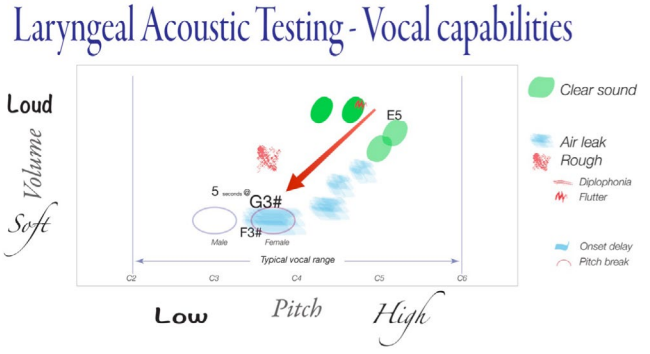
Low pitch, low volume leak, clear high pitch/vol.

Bowing



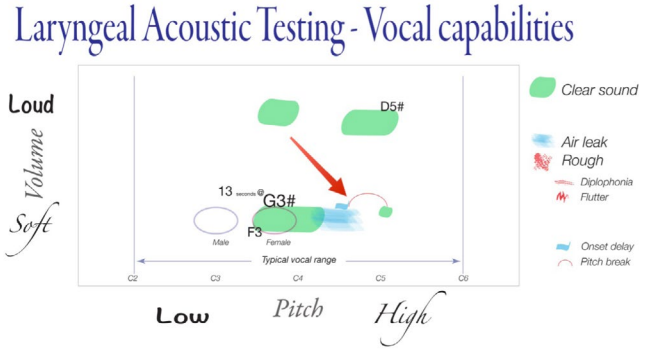
Low pitch, any volume region

Paresis LCA



Low pitch, soft volume region

Central swellings



High pitch, soft volume region

These patterns are the vocal signature for a given type of vocal impairment. Each pattern predicts what pathology will be seen during laryngoscopy and it suggests to the examiner what pitch and volume combinations are most likely to reveal the pathology to the examiner during laryngoscopy. The red arrows point toward the region of maximal vocal abnormal findings and in recurrent laryngeal nerve paresis, that is usually the low pitch, low volume area where there is no compensation from the cricothyroid muscle. Bowing resembles this pattern except that high volume, low pitch is typically quite clear. Vocal swellings have an entirely different picture.